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A Contribution to the pathology of laryngeal phthisis.

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A CONTRIBUTION TO THE PATHOLOGY OF LARYNGEAL PHTHISIS.

By Richard Lake, F.R.C.S. Eng.,
late pathologist, registrar, and senior clinical assistant to the hospital for diseases of the throat, golden square, london.

Laryngeal phthisis is acknowledged to be rarely a primary disease, whilst, on the contrary, it is quite a common complication in pulmonary tuberculosis. Regarding its origin in the latter case, auto-infection is freely accepted by all, though there still exist those who hold that the deposits of tubercle bacilli in the laryngeal tissues have been conveyed there chiefly by the blood and lymph streams, as opposed to those who hold this to be the rarer channel of infection as compared with their introduction from the surface, being carried from the lung in the sputum.

There can be little doubt, as far as clinical and pathological evidence shows, that both these channels of infection exist, either acting separately, conjointly, or one following the other.

The shallow tubercular ulcers are the product of surface-infection, and probably that form of disease which starts between the muscular layers and deep to them is caused by the first-mentioned cause. When deep involvement and superficial ulceration are present it is difficult to form an opinion as to whether blood or lymphatic infection is present, or whether the infection is exclusively from the surface.

The points to which I wish to draw attention are chiefly that the surface-infection is caused originally by the micrococci present along with Koch's bacillus in the sputum, and that the ulceration and tubercular infection are secondary to abscess-formation in the epithelium; and incidentally to the question as to whether the bacillus itself often finds its way between the epithelial cells.

To deal with the latter question first, as the evidence is chiefly negative, I quote Fränkel,[1] being, as far as I can find, the only observer who has absolutely demonstrated Koch's bacillus in the inter-epithelial spaces. For my own part, I must say that I have failed to demonstrate tubercle bacilli in the epithelium, even when the corium has been partly destroyed by ulceration, though the greater the depth of the epithelial ulcer apparently the greater is the probability of finding them.

Heinze[2] in his observations on the cadaver most emphatically corrob-

[1] Deutsche med. Wochenschr., 1879, No. 2.
[2] Bosworth: Diseases of Nose and Throat, pp. 608 et seq.

orates the fact that the shallow ulcers are non-tubercular, when he says that out of fifty lesions 83 per cent. were specific and 17 per cent. were not; these latter were shallow ulcers, and no bacilli were found in them.

Whether Heinze himself intends the interpretation here put to his investigations one cannot tell, but it certainly bears that rendering well, and one is justified in suggesting it.

Basworth[1] makes various allusions and remarks on the subject, some of which are repeated here from the direct bearing they have on the subject.

He says, "The theory of Louis, ... that the laryngeal ulceration results from the eroding action of the sputum as it makes its way over the laryngeal tissues from the diseased parts below, is at the present day indorsed by but few observers;" and further on, "that auto-inoculation of the laryngeal membrane is altogether probable; that this should occur, however, it is necessary that the lesion should be the seat of catarrhal erosion." To quote once more from the same author, "the influence therefore of a chronic laryngitis as a predisposing cause of tubercular disease is altogether probably greatly over-estimated, although it is undoubtedly true that a phthisical patient suffering with chronic laryngitis, especially if this has resulted in erosions, is more likely to develop tubercular action than one in whom the laryngeal membrane is in a state of health."

Regarding the theory of Louis one only needs insert the term micro-organism-laden before sputum, and the theory is quite that usually accepted. With the second quotation not many will disagree, though for my own part I must admit that I look upon a catarrhal laryngitis in tubercular disease of the lungs as being a distinct warning to attend to the larynx.

Moritz Schmidt[2] agrees with Schottelius's view, that in hypertrophic conditions of the interarytenoid space the bacillus-laden sputum is retained between the rugæ, and by maceration and irritation causes ulceration, and thus the bacillus finds an entrance into the tissues.

Here, again, is corroborative evidence in favor of a discontinuity of surface being requisite before infection takes place. My own observations quite tally with this—that is to say, ulceration is frequently present at the base of the clefts, whether caused by surface ulceration or by microscopic abscess-formation, I cannot say; probably both, for the epithelium is very thin in this situation when compared with that over the hypertrophies.

From the foregoing *résumé* it is clear that most observers consider destruction of the epithelium one part, if not in its entirety, the first stage to tubercular ulceration in the larynx; the point which now re-

[1] Die Kehlkopfschwindsucht, etc., Leipzig, 1879 (Lennox Browne).
[2] Moritz Schmidt: Die Krankheiten der oberen Luftwegen, p. 289.

mains to arrive at is, how this ulceration takes place, whether by desquamation or otherwise.

A by no means unimportant question here is the "catarrhal" ulcer; firstly, does it exist? One must admit the existence of ulceration, neither tubercular nor syphilitic; but is it catarrhal? If a catarrhal ulcer exists, and comes into existence by simple erosion, always excepting those cases where excretion is pent up in a crevice, then, naturally, nothing further is to be said regarding the ulceration.

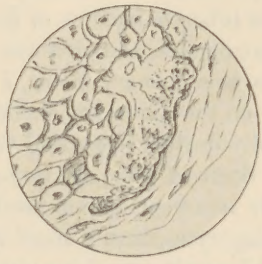
But in none of my sections has any evidence of ulceration thus produced been found, nor has it been proved elsewhere.

In many cases of hypertrophy of the posterior wall of the larynx the masses do not show changes from simple hypertrophy, though pulmonary phthisis is clearly present. The posterior wall is chiefly referred to for the reason that the curette can easily remove hypertrophic masses of tissue here, whereas in other situations the surface only of the ulcer can be scraped off.

FIG. 1.

Three phagocytes in an interpapillary cleft: the phagocytes are full of cocci. × 1/12 oil immersion.

FIG. 2.

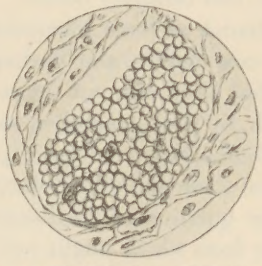


Epithelial abscess just below the surface full of detritus. × 1/12 oil immersion.

FIG. 3.

The crosses (×) are in small abscess-cavities containing cocci.

FIG. 4.



Large epithelial abscess full of pus.

In a considerable number of these little masses I have found a certain fairly uniform condition, viz., the submucous tissue is practically healthy but in the epithelial layer there is a tendency to abscess-formation. These tiny abscesses are filled with leucocytes which have found their way up through the lower layers of epithelium, Fig. 4, and as the

abscess, either by reason of normal desquamation or normal epithelial growth, approaches the surface, the leucocytes become converted into granular detritus, Figs. 2 and 3. In Fig. 1 is shown a papilla in which are seen three phagocytes conveying cocci from the surface.

In none of the epithelial abscesses have I seen a single tubercle bacillus, but I have found cocci in some. It has been shown by a large number of observers that the parts most favorable for the development of tubercular ulceration are those covered with squamous and not ciliated epithelium. This is doubtless perfectly true, but it has no actual bearing here, for, whereas it was accepted for bacilli, it must be admitted for cocci and bacilli together.

So I would put forward the following as the probable processes or stages through which the affection of the mucosa passes from health to tuberculous ulceration. Before doing so, I would say that in pulmonary tuberculosis in which there are no other micro-organisms present these arguments would not hold good.

Also, I would draw attention to the experiments which go to prove that the tubercle bacillus of Koch does not of itself cause abscess-formation, but requires the presence of cocci.[1]

Thus, as a primary factor, I would suggest that the micrococci being at rest on the epithelial surface between the attacks of coughing, and especially during sleep, find their way into the interstices between the cells. Some of these are removed by phagocytes; others are, by reason of their numbers, able to establish themselves, and, by destroying cells and phagocytes, form a small abscess. The abscess ruptures, and is still, bacteriologically speaking, non-tubercular. The ulceration continues, and as the abscess increases in depth the risk of infection by Koch's bacillus increases, and by the time submucous tissue is reached, if not earlier, this certainly occurs.

Inasmuch as these ulcerations take place very frequently in the posterior commissure, and perhaps more frequently there than elsewhere, and that they also are more frequent when hypertrophy is present, catarrh, by producing them as well as a general thickening of laryngeal epithelium, must predispose to infection.

From these and other observations it seems clear that when considering the question of treatment, whatever method is adopted, there are certain facts which might well be borne in mind, viz.:

Ulceration is not the first symptom of laryngeal invasion. The presence of other micro-organisms in the sputum probably increases the risk of laryngeal invasion.

If by intertracheal medication the bacteria can be rendered harmless, the larynx will be favorably influenced. In superficial ulceration

[1] Strauss: Sem. Méd., May 10, 1894; N. Y. Med. Journ., July 7, 1894.

caustic applications, viz., chromic acid 3 per cent., antiseptics and lactic acid, are all equally efficacious.

As assistant in Dr. Bond's clinic at Golden Square, I have had a very fair number of these cases, the custom being to follow Heryng's treatment, locally, with internal administration of creosote; but when there was nothing but simple superficial ulceration, 3 per cent. solution of chromic acid carefully applied yielded excellent results, and this is not to be wondered at, for it hardens the membrane to act as a protective investment. Thyroid extract, so beneficial in lupus, yielded no results in any cases so treated in the forementioned clinic.

Harley St., London, W.